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ERYTHROMYCIN ALTERS PSEUDOMONAS AERUGINOSA ADHERENCE TO BASEMENT MEMBRANE COLLAGEN AND MORPHOLOGY IN VITRO

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Pseudomonas aeruginosa (PA) is a Gram -ve bacilli which colonises the airways of patients with bronchiectasis and cystic fibrosis and causes immense morbidity. Low dose erythromycin (EM) is effective in treating diffuse panbronchiolitis when PA infection is common although the underlying mechanism for this efficacy is unknown. PA adheres to basement membrane avidly *in vivo* and *in vitro*. We have therefore developed a model to determine if PA adherence to basement collagen type IV is affected by the presence of low dose EM. We used scanning electron microscopy (SEM) to measure PA adherence to collagen surface (coated on an inert surface) as the mean number of PA/20 random SEM fields (4000x) to collagen after incubation in 37°C for 45 min. PA was cultured overnight in brain heart infusion before adherence experiments. PBS re-suspended PA (n=12) had mean±SE inocular size of 5.1±2.0, 4.2±1.3, 10.3±4.7, and 6.9±3.5 x10^9 PA/ml after 24h incubation in 0, 5, 0.5, 0.05 mg/ml of EM. Mean (± SD) PA adherence density (20 SEM fields/log inocular size) for PA obtained from 0 µg/ml EM was 56.8±12.5 which was significantly ($p<0.05$) higher than PA obtained from 5, 0.5, and 0.05 µg/ml of EM (21.1±3.5, 23.3±4.2, and 21.5±5.1 respectively). In the absence of EM, the mean surface area and volume of PA bacilli were 2.5±0 & 0.3±0 which were significantly ($p<0.05$) larger than in the presence of 0.05 (2.1±0, 0.2±0), 0.5 (2.0±0, 0.2±0), and 5 ug/ml EM (2.3±0 units, 0.3±0 units) respectively. Our results show that EM reduces PA adherence to human basement membrane collagen and alters PA surface area and volume. These in vitro findings could help explain the clinical efficacy of low dose erythromycin in bronchiectasis.

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GASTRO-OESOPHAGEAL REFLUX IN BRONCHIECTASIS

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Gastro-oesophageal reflux (GOR) and micro-aspiration of gastric acid into the lower respiratory tract could is a recognized cause of bronchiectasis although this has not been studied in a systematic manner. Bronchiectatic patients suffer from chronic sputum production, often as a result of idiopathic inflammation and infection in the tracheobronchial tree. The source of this inflammation is largely unknown although it is mediated by pro-inflammatory mediators. We have performed this systematic study to evaluate GOR in 26 patients (18F; mean age 55.4; mean FEV_1/FVC=1.0/1.71) with stable bronchiectasis but no upper gastro-intestinal symptoms. We have also determined sputum leukocyte density, 24 volume, and pathogen densities simultaneously. A two channel Altimony esophageal pH catheter was passed into the esophagus nasally and kept *in situ* for 24h. The upper channel, situated at the upper esophagus, recorded a mean (±SD) of 30.7±46.7, 0.7±1.1, 21.7±28.3min, 1.5±2.0%, and 7.9±8.9 as the no. of reflux episodes (RE), no. of reflux>5min, time pH<4, fraction time pH<4, and DeeMeester Score respectively. For the lower esophageal channel, these were 59.3±85.5, 3.9±8.7, 88.2±164.6, 6.2±11.6 and 23.2±41.8 respectively. For the upper channel, the fraction time pH<4 correlated with 24h sputum volume ($r=0.73$, $p=0.000$), and reflux >5 min correlated with sputum leukocyte density ($r=0.5$, $p=0.02$). The DeeMeester score of the upper channel showed correlation with 24 sputum volume ($r=0.52$, $p=0.01$) and leukocyte density ($r=0.49$, $p=0.02$). There was no correlation between the lower esophageal recordings with the aforementioned parameters. Our results GOR might play a significant pathogenic role in the pro-inflammatory process in bronchiectasis. Future studies should be pursued in these directions to evaluate this phenomenon further.